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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
TURNER, SHARON L	

ART UNIT	PAPER NUMBER
1647	

DATE MAILED: 09/30/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/316,387	SOLOMON ET AL.	
	Examiner	Art Unit	
	Sharon L. Turner	1647	

-- Th MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *27 June 2003* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *24 and 28-49* is/are pending in the application.

4a) Of the above claim(s) *28 and 36* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *24,29-35 and 37-49* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) 6-27-03
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

Continued Prosecution Application

1. The request filed on 4-23-03 for a Continued Prosecution Application (CPA) under 37 CFR 1.53(d) based on parent Application No. 09/316,387 is acceptable and a CPA has been established. An action on the CPA follows.

2. The Examiner acknowledges the communication of 4-23-03 and 6-27-03 including IDS submission and 37 CFR 1.132 Declaration. The IDS and declaration are duplicates of the submissions of 10-23-02. The IDS and declaration have been entered into the record and have been fully considered by the Examiner. It is noted that reference QR contains the typographical error "2997" as the year of publication. The IDS has been corrected to reference 1997. The submission of 6-27-03 was transmitted a second time on 7-10-03 via Fax due to it's apparent loss while the file was being scanned to the "Image File Wrapper" system. It is thus noted that the 6-27-03 and 7-10-03 submissions in IFW are duplicates.

3. Claims 10-23 and 25-27 have been canceled via the amendement of 6-27-03.

4. Claims 24, and 28-49 are pending. It is noted that this differs from Applicant's summary.

Election/Restriction

5. Applicant's election with traverse of species monoclonal antibodies reactive with a non-light chain amyloid, identified by applicants as claims 23-36 and 39-45 in Paper No. 14 (2-4-02) is acknowledged. The traversal is on the ground(s) that the Office Action fails to provide evidence of any significant search burden with respect to the delineated species. This is not found persuasive because as set forth the species are

patentably distinct as they lack a common core structure and differ in functional properties with different use, different modes of operation, different functions and different effects. Thus a search for any one of the species would not reveal all pertinent art to any other species and thus the search and examination of all species in a single application may place an undue burden upon the Examiner.

The requirement is still deemed proper and is therefore made FINAL.

6. As set forth in Paper No. 15, mailed 4-23-02, newly submitted claims 28 and 36 are directed to an invention that is independent or distinct from the invention originally claimed for the following reasons: Applicants have elected species of monoclonal antibodies reactive with a non-light chain amyloid. Applicants have identified the elected invention as readable on claims 28 and 36. However, claims 28 and 36 are drawn to antibodies raised against immunoglobulin light chain and to monoclonal antibodies reactive with immunoglobulin light chains and thus are directed to non-elected species.

Since applicant has received an action on the merits for the originally presented invention, this invention has been constructively elected by original presentation for prosecution on the merits. Accordingly, claims 28 and 36 are withdrawn from consideration as being directed to a non-elected invention. See 37 CFR 1.142(b) and MPEP § 821.03.

Claim Rejections - 35 USC § 102

7. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

8. Claims 24, 29-31, 35, 39-46, 48-49 are rejected under 35 U.S.C. 102(b) as being anticipated by Konig et al., WO96/25435, 22 August 1996

Konig et al., teach methods of diagnosis, screening and therapeutics for treating unique forms of amyloid peptide deposition including Alzheimer's disease using antibody administration, see in particular p. 6 lines 1-9, 21-22, p. 7, lines 21-27, p. 14, lines 9-11, and p. 25, lines 14-17. The embodiments include all immunoglogical and related methodologies applied to the detection, monitoring, extraction, inhibition and modification of beta-amyloid species in the diagnosis and treatment of Alzheimer's disease, see in particular p. 25, lines 14-17. Konig et al., teach administration of monoclonal antibodies which bind amyloid fibrils, see in particular claims 9-20 and pp. 6-8, including for treatment of Alzheimer's disease. The antibodies specifically bind amyloid fibrils, see in particular p. 6, line 25. Thus, the reference teaches a method which comprises administration of beta amyoid antibodies (immunoglobulin polypeptides) for extraction (removal) of amyloid beta peptides and the treatment of Alzheimer's disease. Claims 15-16 further teach methods of preventing aggregation of beta amyloid peptide comprising administering monoclonal antibody reactive with beta amyloid peptides as in claims 1 and 5. While the reference does not explicitly teach administration "in a patient" as recited in the claim. The artisan well recognizes that such is clearly implied by the teachings directed to therapeutic treatments of

Alzheimer's disease comprising administration of the noted antibodies. In particular, Alzheimer's disease is known to be an etiology that affects human patients, particularly the elderly via amyloid plaque deposition and accumulation within the brain, see in particular Background, pp. 1-5. Konig further teaches that the antibody treatment is effective in a method for the prevention of aggregation of beta amyloid peptide by administration of the antibody, see in particular p. 7, lines 21-23, p. 13, lines 17-20, p.14, lines 6-11 and p. 25, lines 14-18, including in the extraction of beta amyloid species. As the antibodies bind beta amyloid as taught by Konig and are effective in therapeutic treatments of Alzheimer's, they are inherently effective to anticipate the claims. The antibodies can be provided in sterile saline or a pharmaceutically acceptable carrier such as Keyhole Limpet Hemocyanin, see in particular p. 17. The antibodies include human and may be labeled by biotinylation or with radioactive tags such as 35S-Met, see in particular p.22-24. Konig further notes at p. 5-7 suitable cross-isotype and cross reactive antibodies and epitopes via various modifications in peptide immunogen and whether the antibody is for example monoclonal or polyclonal. Specific embodiments of monoclonals are disclosed from p. 19-23.

Applicant's argue that Konig merely teaches methods of generating antibodies and methods of using the antibodies to detect amyloid in post-mortem tissue and only shows the results of immunohistochemical studies. Applicant's argue that Konig does not teach administration of the antibodies to a patient for any reason, let alone to remove amyloid deposits. Applicant's argue that Konig does not show that their antibodies are able to remove amyloid deposits from a patient. Applicant's additionally

argue in the Biere declaration that the usefulness of antibodies as diagnostics and in binding and detecting amyloid plaques does not suggest that the antibodies are effective in removing amyloid plaques from a patient and that the reference therefore does not anticipate the claimed invention.

Applicant's arguments have been fully considered but are not persuasive. First, Konig does teach methods of genereating antibodies and methods of using the antibodies to detect amyloid in post-mortem tissue using immunohistochemical studies. Yet, Konig et al also teach the use of the antibodies in methods of treatment for Alzheimer's disease including for extraction of beta amyloid species and in therapeutic compositions for the treatment of Alzheimer's disease.. In contrast to Applicant's interpretation, Konig teaches the administration of the antibodies to Alzheimer's patents which are known to be human patient subjects. It is true that Konig does not teach the definitive mechanism for the treatment provided to Alzheimer's patients via administration of antibodies to beta-amyloid. However, the mechanism of the treatment is not required for Konig to be enabling. Moreover, Konig does allude to the mechanism in part by their reference to extraction of beta amyloid species. It is well accepted in patent law that a newly discovered property does not make a compound or method newly patentable. Similarly here, the claiming of an old method via the use of mechanistic limitations such as the recitation of "an amount effective to remove amyloid deposits" does not evidence patentability over the prior art teachings. The question is whether or not the methods are the same or different. In this analysis both methods provide the same antibodies to the same patients for the same purpose. Moreover,

Konig notes that their method is effective for treatment and thus the mechanism is inherently provided absent convincing factual evidence to the contrary. It is Applicant's burden to show unobvious difference as the PTO has insufficient resources to compare the teachings of the prior art reference and that of Applicant's claims. Reasons showing inherency have clearly been shown and there are no limitations to the antibodies, their amounts or routes of administration that would teach over the prior art reference. Thus, the reference teachings anticipate the claimed invention.

9. Claims 24, 29-35 and 37-49 are rejected under 35 U.S.C. 102(b) as being anticipated by Nettleship et al., EP613007, 8-31-1994.

Nettleship et al., teach antibodies useful in the diagnosis and treatment of mammals suffering from Alzheimer's Disease, see in particular column 7, line 39-column 8, line 18. The antibodies are beta-amyloid peptides, particularly in beta-sheet conformation, but also include antibodies to alternative fragments, see in particular column 1, line 52-column 2, line 56. It is understood that the functional embodiment which characterizes the diagnostic and therapeutic relationship as disclosed in Nettleship hinges on the binding of the antibodies to the beta-amyloid peptides, see in particular reference in paragraph spanning columns 7-8 and reference to numerous assay systems suitable to detect agents which bind, column 8, lines 6-15. In addition, the compositions are pharmaceutical compositions which include formulations for parenteral administration (other than by intestinal, i.e., subcutaneous, intravenous, etc., as understood by the skilled artisan), see in particular column 8, lines 19-42. .

Nettleship et al., teach the use of alternatively produced beta amyloid antibodies including to peptides which have adopted a random coil or alpha-helix conformation and to antibodies which are genetically engineered, antibody fragments, chimeric antibodies, recombinantly produced antibodies, and "humanized or murinized" antibodies as generated by replacement of CDR regions, see in particular column 5, lines 42-column 6, line 20. Thus the reference teaches the variable or cross-reactive antibodies of the claims. It is noted that the polyclonal sera would inherently include multiple antibodies and Ig isotypes. It is further noted that the patient population includes mammals and thus encompasses humans and human antibodies, particularly of Alzheimer's patients, see in particular columns 6-7. Further the reference is effective in the treatment of Alzheimer's. Thus, the reference is to be enabling for the determination of appropriate doses and routes of administration suitable for treatment. The mechanism whereby the treatment occurs, via removal of amyloid, is inherently is provided.

Similar to Konig, it is true that Nettleship does not teach the definitive mechanism for the treatment provided to Alzheimer's patients via administration of antibodies to beta-amyloid. However, the mechanism of the treatment is not required for Nettleship to be enabling. It is well accepted in patent law that a newly discovered property does not make a compound or method newly patentable. Similarly here, the claiming of an old method via the use of mechanistic limitations such as by the recitation of "an amount effective to remove amyloid deposits" does not evidence patentability over the prior art teachings. The question is whether or not the methods are the same or different. In this analysis both methods provide the same antibodies to the same patients for the

same purpose. Moreover, Nettleship notes that their method is effective for treatment and thus the mechanism is inherently provided absent convincing factual evidence to the contrary. It is Applicant's burden to show unobvious difference as the PTO has insufficient resources to compare the teachings of the prior art reference and that of Applicant's claims. Reasons showing inherency have clearly been shown and there are no limitations to the antibodies, their amounts or routes of administration that would teach over the prior art reference. Thus, the reference teachings anticipate the claimed invention.

Claim Rejections - 35 USC § 103

10. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103© and potential 35 U.S.C. 102(f) or (g) prior

art under 35 U.S.C. 103(a).

11. Claims 24, 29-35 and 37-49 are rejected under 35 U.S.C. 103(a) as being unpatentable over Konig et al., WO96/25435, 22 August 1996, Nettleship et al., EP613007, 8-31-1994 and Immunology: a short course, Benjamini & Leskowitz Ed., Wiley-Liss, Inc., New York, NY, page 142.

Konig et al., teach as set forth above. In particular Konig teaches methods of diagnosis, screening and therapeutics for treating unique forms of amyloid peptide deposition using antibodies. Konig et al., teach administration of monoclonal antibodies which bind amyloid fibrils, see in particular claims 9-20 and pp. 6-8 for treatment of Alzheimer's disease. The antibodies specifically bind amyloid fibrils, see in particular p. 6, line 25. Thus, the reference teaches a method which comprises treating a patient having an amyloid deposition disease by administration of an immunoglobulin polypeptide which binds to an amyloid fibril. Konig further teaches that the antibody treatment is effective in a method for the prevention of aggregation of beta amyloid peptide by administration of the antibody, see in particular p. 7, lines 21-23, p. 13, lines 17-20, p.14, lines 6-11 and p. 25, lines 14-18. The antibodies can be provided in sterile saline or a pharmaceutically acceptable carrier such as Keyhole Limpet Hemocyanin, see in particular p. 17. The antibody intrinsically opsonizes upon binding, as evidenced by Benjamini et al., which teach at p. 142 that IgG immunoglobulin antibodies bind and mediate opsonization or removal via phagocytosis. Thus, the artisan would equate the mechanistic recitation of inhibiting formation, removal or modulation of amyloid deposition as being achieved. The antibodies may be labeled by biotinylation or with

radioactive tags such as 35S-Met, see in particular p.22. Konig further notes at p. 5-7 suitable cross-reactive antibodies and epitopes for various modifications. Specific embodiments of monoclonals are disclosed from p. 19-23.

Nettleship et al., teach as set forth above. In particular Nettleship teaches antibodies useful in the diagnosis and treatment of mammals suffering from Alzheimer's Disease, see in particular column 7, line 39- column 8, line 18. The antibodies are beta-amyloid peptides, particularly in beta-sheet conformation, but also include antibodies to alternative fragments, see in particular column 1, line 52-column 2, line 56. It is understood that the functional embodiment which characterizes the diagnostic and therapeutic relationship as disclosed in Nettleship hinges on the binding of the antibodies to the beta-amyloid peptides, see in particular reference in paragraph spanning columns 7-8 and reference to numerous assay systems suitable to detect agents which bind, column 8, lines 6-15. In addition, the compositions are pharmaceutical compositions which include formulations for parenteral administration (other than by intestinal, i.e., subcutaneous, intravenous, etc., as understood by the skilled artisan), see in particular column 8, lines 19-42. Thus, the reference appears to be enabling for the determination of appropriate doses and routes of administration suitable for such binding, inhibition of formation, removal or modulation of amyloid deposition to occur. Nettleship et al., teach the use of alternatively produced A antibodies including to peptides which have adopted a random coil or alpha-helix conformation and to antibodies which are genetically engineered, antibody fragments, chimeric antibodies, recombinantly produced antibodies, and "humanized or murinized"

antibodies as generated by replacement of CDR regions, see in particular column 5, lines 42-column 6, line 20. Thus the reference teaches the variable or cross reactive antibodies of the claims. It is noted that the polyclonal sera would inherently include multiple antibodies and Ig isotypes. It is further noted that the patient population includes mammals and thus encompass humans and human antibodies, see in particular columns 6-7.

Applicants specification at pp. 14-16 also teach the routine of one of skill in the art to produce humanized and chimeric antibodies.

Neither Konig nor Nettleship specifically teach the mechanistic effects of antibody administration as recited in the claims, i.e., the inhibition of formation, removing amyloid deposits or the modulation of formation of amyloid deposits. However, Konig et al., teaches that antibody administration is effective to prevent aggregation or for extraction of amyloid deposits and Walker and Nettleship teach the use of antibody administration for the treatment and prevention of Alzheimer's Disease mediated via preventing and treating amyloid deposition. However, Benjamini et al., teach as recognized in the art that antibody binding mediates opsonization and removal of IgG bound material in the host.

Thus, it would have been prima facie obvious to the skilled artisan to utilize either the antibodies of either Konig or Nettleship for the in vivo administration and treatment of patients, particularly with Alzheimer's Disease. Further it would have been prima facie obvious based on the teachings of Konig, Nettleship and Benjamini that such treatment is effective to remove beta-amyloid thus treating Alzheimer's via modulating

the levels of amyloid in patients. One of skill in the art would have been motivated to provide such a method based on the cumulative reference teachings and the recognition in the art of opsonization based upon antibody binding to beta-amyloid. The effective amounts are provided by the antibody compositions effective for binding and routing. Further, one skilled in the art would have expected success using such a method based upon the high skill in the art of antibody technology and the combined teachings of Konig, Nettleship and Benjamini in the treatment of amyloid deposition disease with non-light chain, beta-amyloid antibody, particularly in Alzheimer's disease. Thus, for the aforementioned reasons, the claimed invention is rendered obvious to the skilled artisan.

Applicant's argue that the Konig reference does not disclose a method of administering antibodies to a patient to remove amyloid deposits from the patient and that according to the declaration of Dr. Biere antibodies used as diagnostics are not suggestive of effectiveness in removing amyloid from a patient. Applicant's argue that the 369.2B antibody was not tested for in vivo administration and that it's use would not be predicted to remove amyloid in an in vivo system. Applicants argue that Becker (Nettleship) does not teach a method of treatment comprising administering antibodies to patients to remove amyloid deposits and the discussion is hypothetically of therapeurtic purposes. Applicant's argue that it was not predictable that the antibody would be effective in removing amyloid deposits.

Applicant's arguments filed 6-27-03 have been fully considered but are not persuasive. In particular, the Examiner notes that the Konig and Becker (Nettleship)

references each teach the administration of beta amyloid antibodies for therapeutic use in Alzheimers treatment. Further, the Benjamini supports the artisan's knowledge of opsonization and removal of material via antibody IgG binding within the host. The references each evidence binding specificity. It is further noted that the mechanism by which the methods effect their treatment is unimportant. The similarity or difference as to the method steps is. In instant case, the steps of the methods within the prior art and instant claims appear identical in that the same antibodies are provided to the same patient population for the same purpose/utility, i.e., the treatment of Alzheimer's as explicitly stated in both prior art references. Moreover, there are no limitations within instant claims as to the route, quantity, type of antibody or otherwise that would indicate any difference in the abilitly of particular antibodies to be successful or not in the claimed method. In short, both the prior art and instant claims evidence the applicability of any antibody in the method to the extent that the antibodies bind to an amyloid fibril or component or precursor thereof. Thus, the cumulative reference teachings render the invention obvious to the artisan.

Declaration of Dr. Biere

12. The declaration under 37 CFR 1.132 by Dr. Biere has been fully considered but is not persuasive. Dr. Biere notes that the focus of amyloidosis research at the time of the invention relates to inhibiting production or enhancing clearance of the precursor protein and not to therapy via the use of antibodies in removal. Dr. Biere further suggests that the successful use of antibodies to clear amyloid was unexpected. Dr. Biere further notes that the binding of antibodies is not predictive of effector function.

The declaration has been fully considered but is not persuasive. The relevant issue is whether or not there is unobvious difference between the prior art teachings and Applicant's claims. The prior art teachings are directed to the invention of administration of beta amyloid antibodies for the treatment of Alzheimer's disease. Both prior art references and applicant's claims indicate that any antibody that binds amyloid is effective and useful in the treatment. There is no evidence or limitations in the claims or specification that indicate that anything other than binding is required such that removal and/or opsonization occurs. In fact, Applicants claims are structured such that any antibody that binds is capable of removal. Moreover as Benjamini teaches removal and opsonization are recognized as one in the same, no differences can be discerned. The declaration provides no evidence as to a direct comparison or to unobvious difference in the claimed methods in comparison to the prior art. Thus, the declaration is ineffective to overcome the prior art of record. The recitations as to mechanisms of the prior art references fails to distinguish over the prior art.

Status of Claims

13. No claims are allowed.

Conclusion

14. Any inquiry of a general nature or relating to the status of this general application should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Papers relating to this application may be submitted to Technology Center 1600, Group 1640 by facsimile transmission. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). Should applicant wish to FAX a response, the current FAX number for Group 1600 is (703) 308-4242.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sharon L. Turner, Ph.D. whose telephone number is (703) 308-0056. The examiner can normally be reached on Monday-Thursday from 8:00 AM to 6:30 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Kunz, can be reached at (703) 308-4623.

Sharon L. Turner, Ph.D.
9/29/03

GARY KUNZ
SUPERVISORY PATENT EXAMINER
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